specification as filed, and Applicants believe that no new matter has been added. Applicants respectfully request that the Examiner reconsider and withdraw the various grounds of rejection of the claims.

I. Rejections Under 35 § 112, first paragraph

Claims 29-32 and 35-37 are rejected under 35 U.S.C 112, first paragraph as being indefinite for failing to point out and distinctly claim the subject matter that Applicants regard as the invention. Specifically, the Examiner states that the metes and bounds of Claims 29, 35, and 37 are vague and indefinite for reciting "codon 531" and for failing to designate the codon which is to serve as the termination or stop codon. Applicants have amended claim 29 upon which claims 30-34 depend, and claim 35 upon which claims 36-37 depend to more distinctly define the claimed invention. As amended Claim 29 is directed to a polynucleotide wherein codon 531 serves as the termination or stop codon. As amended Claim 35 is directed to an oligonucleotide that is capable of recognizing and distinguishing a mutant cSrc where codon 531 of the mutant serves as the termination or stop codon.

Applicants believe that these amendments address the Examiner's concerns under 35 U.S.C. 112, first paragraph. Reconsideration of the rejection and withdrawal thereof is respectfully requested.

II. Rejections Under 35 § 102

On page 4 of the Office Action the Examiner rejected claims 29-36 and 38 as anticipated by U.S. Patent No. 5,336,615. Specifically the Examiner states that the '615 Patent teaches an isolated polynucleotide encoding a mutant c-Src protein wherein the polynucleotide has a codon 531 which is a stop codon comprising SEQ ID NO: 1.

Applicants respectfully traverse. Applicants submit that the '615 Patent is not analogous art. The '615 Patent discloses genetically engineered endothelial cells having enhanced migration and enhanced plasminogen activation activity. In contradistinction, the present inventors have surprisingly discovered that a novel mutation at Src 531 is responsible for malignant transformation and metastasis. Thus, the present inventors disclose a mutant form c-Src that plays a role in Src activation of cancer. This role, indeed this entire field of study is not

even contemplated in the '615 Patent, which is directed towards cardiovascular health. Thus, Applicants respectfully submit that the '615 Patent should not be treated as prior art with respect to the present invention.

Further, a detailed review of the '615 Patent fails to provide a disclosure of an isolated polynucleotide encoding a mutant c-Src protein, wherein the polynucleotide has a *codon 531 which is a stop codon*. Indeed, the '615 Patent discloses the native c-Src gene; that is, the gene encoding the naturally occurring protein, which does not demonstrate the enhanced activity demonstrated by the mutant cSrc disclosed in the subject application.

Anticipation requires that each and every element of the claimed invention be disclosed in a single reference. Thus, the absence from a reference of any claimed element negates anticipation. In view of these remarks, Applicants maintain that the claims now under consideration are not anticipated by the '615 Patent and respectfully request that the Examiner reconsider and withdraw the rejection under 35 U.S.C. § 102.

III. Rejections Under 35 § 103

On page 5 of the Office Action, the Examiner rejected claims 29-38 under 35 U.S.C. 103(a) as being unpatentable by U.S. Patent Number 5,336,615 stating that one of ordinary skill in the art would have been motivated to place the recited oligonucleotides in a kit.

As stated previously, Applicants do not believe that the '615 Patent is analogous art to the present invention. Further, the test under §103 is whether the claimed invention considered as a whole would have been obvious. The invention as a whole is not restricted to the specific subject matter claimed, but also embraces its properties and the problem it solves. *See, In re Wright,* 6 U.S.P.Q.2d 1959 (Fed. Cir. 1988). Applicants have provided a mutated cSrc useful in the diagnosis, therapy, and prevention of cancer. As mentioned, the '615 Patent is directed toward cardiovascular health. The '615 Patent neither discloses nor suggests truncation of the polynucleotide at codon 531, and the benefits of such a mutant protein in the field of cancer, including diagnosis, therapy, and prevention.

AUTHORIZATION

A Notice of Appeal with appropriate fees is being filed concurrently herewith. In the event that an extension of time is required, or which may be required in addition to that requested in a petition for an extension of time, the Commissioner is requested to grant a petition for that extension of time which is required to make this response timely and is hereby authorized to charge any fee for such an extension of time or credit any overpayment to Deposit Account No. 50-1710.

CONCLUSION

All of the stated grounds of objection and rejection have been properly traversed, accommodated, or rendered moot. Applicants therefore respectfully requests that the Examiner reconsider all presently outstanding objections and rejections and withdraws them. There being no other objections or rejections, Applicant respectfully requests that the present application be allowed and pass to issue.

Respectfully submitted,

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APPENDIX

MARKED-UP VERSION TO SHOW CHANGES MADE

The claims are amended as follows:

29. (Amended) An islolated polynucleotide encoding a mutant c-Src protein, wherein the polynucleotide has a codon 531, and wherein the codon 531 is a stop codon [which is a stop codon].

35. (Amended) An oligonucleotide capable of recognizing and distinguishing a mutant c-Src gene having a codon 531, wherein the codon 531 is a stop codon [which is a stop codon] from a wild-type c-Src gene.